

Are biological neuron that vicious ? Or only their models ?

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Abstract

With care, and keeping in mind that the biological plausibility of neuronal models with respect to the real brain activity is still an open question, we revisit some mathematical and numerical aspects of generalized Integrate and Fire (gIF) models and propose to eliminate assumptions related to spurious discontinuities. This concerns both the fire regime and then the integrate regime of the neuron.

With this new point of view, some “biological” results obtained on “models” are to be reconsidered. This has also positive consequences. It allows us to reduce the bio-physical membrane equation to a very simple but powerful gIF numerical model. This also dramatically reduces the algorithmic complexity of event-based network simulations, as experimented here.

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1 Introduction

Let us consider biological models of cortical maps (Grossberg, 1988; Koch & Segev, 1998; Dayan & Abbott, 2001), in a context where the spiking nature of neurons activity (Gerstner & Kistler, 2002) is made explicit, either from a biological point of view or for computer simulation (Brette et al., 2007). As reviewed recently (Izhikevich, 2004), from the detailed Hodgkin-Huxley model (Hodgkin & Huxley, 1952), (still considered as the reference but unfortunately intractable when considering neural maps), back to the simplest integrated and fire (IF) model (see e.g. (Gerstner & Kistler, 2002)), a large family of continuous-time models have been produced, often compared with respect to their (i) biological plausibility and their (ii) simulation efficiency.

Biological plausibility at the neuron level is understood as the capability to reproduce what is observed at the cell level, often considering in-vitro experiments (Koch & Segev, 1998). This is a questionable point of view as shown in recent experiments in V1 (Frégnac, 2003, 2004) where it appears that a single-cell observation highly differs between in-vitro and in-vivo conditions. Biological plausibility at the neural network level is understood as the capability to reproduce what is observed regarding e.g. the cortical map activity (Carandini et al., 2005). This includes predicting the response not only to *specific artificial* but also *natural* stimuli: this means, for V1, taking natural image sequences input shifted by eye movements into account (Baudot, Marre, Levy, & Frégnac, 2007), after the retinal and LGN processing (see e.g. (Simoncelli & Olshausen, 2001) for a discussion about information processing in these structures).

Considering generalized Integrate and Fire models

At the present state of the art, considering adaptive exponential integrate-and-fire model with conductance based synaptic interaction (as e.g. in (Destexhe, 1997; Brette & Gerstner, 2005; Rudolph & Destexhe, 2007)), called generalized Integrate and Fire (gIF) in the sequel, presents several advantages:

1. Though these models have mainly considered one neuron dynamics they are easy to extend to network structure with synaptic plasticity (STDP, Hebbian learning) (Markram, Lübke, Frotscher, & Sakmann, 1997; Pfister & Gerstner, 2006).
2. They seem to provide an effective description of the neuronal activity allowing to reproduce several important neuronal regimes, with a good adequacy with respect to biological data, especially in high-conductance states, typical of cortical in-vivo activity (Destexhe, Rudolph, & Paré, 2003).
3. They provide nevertheless a simplification of Hodgkin-Huxley models, useful both for mathematical analysis and numerical simulations (Gerstner & Kistler, 2002; Izhikevich, 2003).

Integrate and Fire Neuron models always incorporate two regimes for the neurone membrane potential evolution: the “integrate” regime and the ”fire” regime. Let us very briefly review this framework.

Integrate regime Below some threshold θ , the membrane potential $V(t)$ of the neuron dynamics is driven by an equation of form:

$$C \frac{dV}{dt} + g(V, t, \tilde{\omega}) V = i(V, t, \tilde{\omega}), \quad (1)$$

where C is the membrane capacity, g is the membrane conductance of the

neuron and I is a current.

Here $\tilde{\omega} = \{\dots t_j^n \dots\}$ is the spike-times list, i.e. the spike times of all neurons (say, t_j^n is the n th spike of the neuron of index j). At the network level a notion of “raster plot” is to be introduced (see (Cessac, 2007) for a mathematical definition) which can be avoided here.

The neuron state is thus characterized by its membrane potential. This is the key point here: at this scale of modelization a *punctual* view of each neuron is proposed, in which soma, dendrites and axon electrical states are reduced to a unique variable (see e.g. (Gerstner & Kistler, 2002) for a deeper discussion).

Each quantity θ , g and I could be a function of the potential V itself (non-linearity), of the spikes $\tilde{\omega}$ (network state dependency) and of time (dynamic quantity): this is going to be carefully discussed in the sequel.

The leaky Integrate and Fire (LIF) equation corresponds to the case where the conductance g is constant.

Fire regime When the membrane potential reaches some threshold θ , a change of regime is observed and the neuron “fires a spike”. This is observed in the detailed Hodgkin-Huxley model (Hodgkin & Huxley, 1952), and the simplification of the gIF models is to consider that

- this event is only characterized by its occurrence time t_j^n (neither its magnitude, nor its duration, ..)
- only this time information is sent by a neuron output to another neuron input.

The notion of fire threshold remains an approximation which is not sharply defined in Hodgkin-Huxley or Fitzhug-Nagumo models (more precisely it is not a constant but it depends on the dynamical variables). This is going

to be discussed latter. To analyze the fire regime, however it is going to be sufficient to fix a positive number θ called “the firing threshold of the neurons” and assume that all neurons have the same firing threshold (see (Gerstner & Kistler, 2002) for a formal demonstration that this can be assumed without loss of generality, up to a change of variable).

This simplification excludes several aspects such as transmissions via the neural glia, etc.. However, at the present state of the art, in the cortex, this seems a realistic approximation to encounter for the neuronal activity.

These assumptions being accepted, it appears that authors very often implicitly introduced additional assumptions, mainly regarding *discontinuities* in the model. It has large when not huge consequences at the analytical or numerical level, which do not correspond to the biological reality. The goal of the paper is to discuss and reject such “vicious” properties. And illustrate the related positive consequences.

Let us now informally introduce the main idea.

Introducing discontinuities

In fact, after a spike, it is always assumed in IF models that an *instantaneous* reset of the membrane potential occurs. This is a formal simplification, and it has a general spurious effect: the information theory (e.g. the Shannon theorem, stating that the sampling period must be less than half the period corresponding to the highest signal frequency) is not applicable with unbounded frequencies. From the information theoretic point of view, it is a temptation to relate this spurious property to the *erroneous* fact that the neuronal network information is not bounded. In the biological reality, time synchronization is indeed not instantaneous (action potential time-course, synaptic delays, refractoriness, ..). More than that, these biological temporal limits are very precious quantitative

elements, allowing to bound and estimate the coding capability of the system.

A step further, when considering spike-time dependent plasticity (STDP), the learning rule is almost always simulated with bio-physically implausible jump in the pre-post synaptic time dependency curve (Gerstner & Kistler, 2002; Badoual et al., 2006). If the idea that considering a discontinuity in the membrane potential after the occurrence of a spike is helpful at a certain level of the analysis, it is a simple abstraction of the bio-physical reality. Thus it must be taken into account with care, especially at a theoretical level.

From a numerical point of view, simulation efficiency is directly impacted by these assumptions. Simulation efficiency is a twofold issue of performance and precision (see (Brette et al., 2007) for a recent review). At the network level, especially for a very large network assembly, event-based simulations, in which firing times are not regularly discretized but calculated event by event at the machine precision level, provides (in principle) an unbiased solution (Rochel & Martinez, 2003) for IF neurons, or any other model of neurons, whenever the next spike-time can be computed with an *analytic form*. Note however that the explicit computation of the next spike-time may require approximations and is anyway submitted to numerical round-off errors. The question is thus whether these errors have an effect on the dynamics, since initial condition sensitivity can be very strong.

Furthermore, it has been shown that a regular clock-based discretization of the continuous neural system introduces systematic errors, with drastic consequences at the numerical level, even when considering very small sampling times (Rudolph & Destexhe, 2007). The qualitative reason for this is obvious: current IF models, intrinsically consider an arbitrary discontinuity of the membrane potential, with an unbounded frequency range in the Fourier spectrum (induced by the instantaneous firing). The question whether this is still meaningful for

biological neurons is open.

What is the paper about

With care, and keeping in mind that the biological plausibility of such models with respect to the real brain activity is still an open question, we revisit in details the related mathematical and numerical aspects of gIF models.

In the next sections we are going to discuss the fire regime and then the integrate regime. We are going to show that this allows us to reduce the bio-physical membrane equation and derive an original and very simple gIF numerical model. In the last sections we are going to show two applications of these general ideas, one regarding the neuronal simulation at the neuron level and one regarding event-based simulations algorithmic efficiency at the network level.

2 The “fire regime”

2.1 From instantaneous reset to temporal characteristics

Let us assume in this section θ constant and identical for all neurons. If $V(t_i) \geq \theta$ one says that “neuron fires at time t_i ”. This corresponds to the following procedure. The neuron membrane potential is reset *instantaneously* to some *constant* reset value V_{reset} and a spike is emitted toward post-synaptic neurons.

In mathematical terms firing is often written:

$$V(t_i) = \theta \Rightarrow V(t_i^+) = V_{reset} \tag{2}$$

The notation t_i^+ stands for an arbitrary small increment of t_i and is an attempt to give a mathematical meaning to the word “instantaneous”. More formally, $V(t)$ is the concatenation of two solutions of (1); (i) one before the spike, $t < t_i$, with the final condition $V(t_i) = \theta$ and the other (ii) after the spike

$t > t_i$ with the initial condition $V(t_i) = V_{reset}$. This illustrates how defining an instantaneously reset implies that the membrane potential function lives in a rather large functional space¹.

In any case, from a mathematical (and logical) point of view, there is an evident problem in defining an “instantaneous” firing. The membrane potential cannot be simultaneously equal to θ and to V_{reset} . A possible way to circumvent this problem is to consider that if the membrane potential reaches the threshold value at time t then the integration goes on up to time $t + \epsilon$, where ϵ can be *mathematically* arbitrary small. This is the meaning of t^+ . But, on biological/physical grounds ϵ *cannot* be arbitrary small. Indeed, in real neuron firing occurs within a finite time dt corresponding to the shortest time scale involved in the spike generation.

Furthermore, in models dealing with conductance based differential equations, like Hodgkin-Huxley’s, this time scale must be sufficiently *large* to ensure that the description of ionic channels dynamics (opening and closing) in terms of *probabilities* is valid. Additionally, Hodgkin-Huxley’s equations uses a Markovian approach (master equation) for the dynamics of the well-known “ h, m, n ” (ionic) gates (Hodgkin & Huxley, 1952). This requires that the characteristic time dt is quite a bit larger than the characteristic time of decay for the time correlations between gates activity. Thus, on phenomenological grounds dt cannot be arbitrary small: one can obviously consider mathematically the limit $dt \rightarrow 0$, but then the equations lose their meaning.

At a very concrete level, let us see how this notion of instantaneous reset is contradicted, as summarized in Figure 3.

¹If g and I do not depend on $V, t, \tilde{\omega}$, thus are constant, the reader easily verifies that the solution of (1) and (2) writes:

$$V(t) = V^*(t) - [\theta - V_{reset}] e^{-g/C (t-t_i)} H(t - t_i)$$

writing $V^*(t)$ the solution of (1) without (2). More generally the solution is a distribution.

Reaction time and spike-time precision

In reality, there is a “reaction time” τ of the neuron which corresponds to the time of raise and fall for the membrane potential. One expects to have $dt \ll \tau$. In IF models with instantaneous reset, however, the time reaction τ being considered as instantaneous, one assumes in fact $\tau \ll dt$.

These raise and fall reaction times induce another fact: there is an intrinsic lower bound for the spike-time precision $\delta\tau$. Considering that the spike-time is defined by the membrane potential maxima $V(t_i)$ at time t_i , we obtain around t_i , assuming differentiability of V :

$$V(t) = V(t_i) + \kappa(t - t_i)^2 + o(|t - t_i|^3),$$

with $\kappa = d^2V/dt^2(t_i)$ and easily derive, as a rule of thumb:

$$\delta\tau \simeq \sqrt{\frac{\langle \delta V \rangle}{\langle \kappa \rangle}}.$$

where the average $\langle \rangle$ is to be taken over a set of measurements.

In order to roughly estimate this characteristic time we have taken a few dozen of spike profiles in several spike trains (Carandini & Ferster, 2000; Koch, 1999) and graphically estimate the values on a zoom of the provided figures. We have obtained $\delta\tau = 0.1ms$, with a peak curvature order of magnitude of $\langle \kappa \rangle = 100mV/ms^2$ as illustrated in Figure 1, considering a voltage precision of $\langle \delta V \rangle = 10\mu V$, i.e. at the order of magnitude of the membrane potential noise (Koch, 1999). Similar numerical values are obtained reading other electrophysiological data (Carandini & Ferster, 2000). No mistake, these numbers simply fix the order of magnitude of a lower bound.

Furthermore, similar order of magnitude is obtained considering the numerical precision in inter-neuron synchronization (Crook, Ermentrout, & Bower, 1998): synchronicity is numerically defined up to $\delta\tau$. This has the following precise meaning: two neurons are not synchronized if one neuron spikes while the other neuron does not spike in a time window of width $\delta\tau$. On the contrary,

they are synchronized if and only if they repetitively both spike in the same time window (indeed if they only spike once or a few times in the same $\delta\tau$ time window this has no statistical significance).

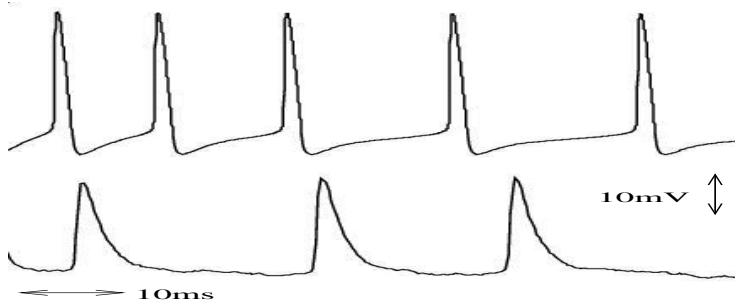


Figure 1: Two examples of spike profiles in the cat primary visual cortex. The peak curvature order of magnitude are $30 - 100mV/ms^2$.

An important example of consequence is related to spike-time dependent plasticity: the usual STDP rule introduces a jump in the pre-post synaptic time dependency curve (Gerstner & Kistler, 2002; Badoual et al., 2006), as schematized in Figure 2, from (Markram et al., 1997; Badoual et al., 2006). This means that if the pre-synaptic spike time t_- is either smaller or larger than the post-synaptic spike time t_+ there is a completely different behavior. This is implausible since as soon as $|t_- - t_+| < \delta\tau$ their order of occurrence is meaningless. The spike-time difference value is ill-defined. and seems to introduce spurious phenomena (Rudolph & Destexhe, 2007). Smoother models such as (Pfister & Gerstner, 2006) do not introduce such a discontinuity and better explain even rather complex STDP phenomena. Considering a probabilistic model for instance, with an additional noise on the observed spike time, obviously yields to a smooth connection between the potentiation and depression, as illustrated in Figure 2, numerical values from (Badoual et al., 2006). This is going to be developed in a near future.

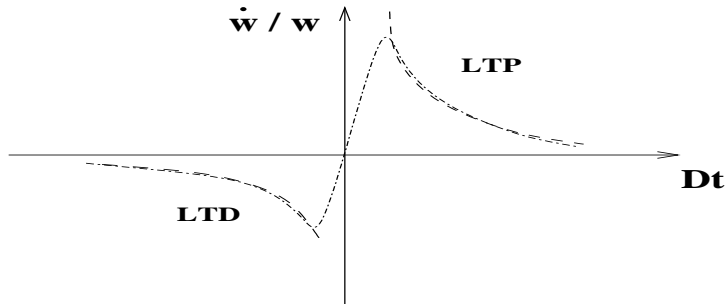


Figure 2: A schematic view of spike-time dependent plasticity. The relative synaptic weight variation \dot{w}/w is positive (potentiation) if the post-synaptic spike t_+ follows the pre-synaptic weight t_- and it has been experimentally observed, for two spikes, that $\dot{w}/w \simeq 0.86 e^{-\frac{t_+ - t_-}{19ms}}$. The relative synaptic weight variation \dot{w}/w is negative (depression) if the pre-synaptic spike t_- follows post-synaptic weight t_+ and it has been experimentally observed, for two spikes, that $\dot{w}/w \simeq -0.25 e^{-\frac{t_- - t_+}{34ms}}$. What occurs when $t_- \simeq t_+$ should correspond to a smooth connection between the two curves, not a jump.

Minimal transmission delays

Another important characteristic time, often “neglected” in abstract models because of the related mathematical complexity, is the neuron to neuron transmission delay δt . Such delays are very small in cortical column but *not negligible*.

The smaller delays (Koch, 1999; Burnod, 1993) involving the pre-synaptic axonal delay, synaptic delay and post-synaptic dendritic delay seems to be at least of $0.5ms$, with delay up to $10ms$ for inter cortical maps transmissions. A step further, many local inter-neuronal connections in the cortex are realized through electrical gap junctions (Galarreta & Hestrin, 2001), this being predominant between cells of the same sub-population (Amitai et al., 2002). In such a case the inter-neuron delays are much smaller, but still measurable, since the transmission is mainly due to the spike potential raise, whose time constant is about $0.1 - 0.2ms$ (see (Lewis & Rinzel, 2003) for a discussion about the electrical transmission in this case). It is thus a reasonable assumption to consider that local electrical connections are delayed by $\delta t \gtrsim 0.1ms$.

Gap junctions delays are much smaller ($\delta t > \simeq 10\mu s$) but still non negligible, as taken into account in the sequel (Koch, 1999).

This has an important consequence: neurons always “spike one after another” in the sense that, a spike does not “immediately” (i.e. with a delay smaller than the spike precision) induces a neighbor neuron spike. If a neighbor neuron spike is synchronized, this comes from a previous cause. This thus prevent the system from any “instantaneous avalanche” and avoid any problem of temporal causality (Rochel & Martinez, 2003). This remark is going to change the performances of a certain class of neural networks simulation as developed in section 6.

Refractory period and maximal spike-time intervals

Another important bound is the refractory period r . After a spike (see e.g. (Koch, 1999)), no subsequent spike can occur during a period $r \simeq 1ms$. Authors consider both an:

- “absolute refractory period” where the membrane potential is maintained close to the reset value while incoming spike are “lost” (i.e. not taken into account), and a
- “relative refractory period”, often following the absolute refractory period, where the potential can still be increased by incoming spikes, but with some “penalty”.

In any case, since the observed spike rate is not higher than $1KHz$ this allows us to bound the spike-time interval, and guarantees that a neuron never spikes more than once in a r interval. This is a very precious result used both at the analytical and numerical level.

Let us fix another bound, less often made explicit in the literature, although always present. Given an isolated neuron, i.e. after the last arrival of a pre-

synaptic spike, there is a maximal delay ΔT after which the neuron has either already fired a spike or will never spike. In other words, we consider here that during the time when it is isolated, a neuron cannot wait an unbounded period of time and then spike. The physical reason is obvious: since there is a leak, the membrane potential decreases with time and thus will never “unexpectedly” raise again, except if the input current balance the conductance leak². Such a singular condition is not generic because the current is generated by a discrete set of synapses thus introducing not a continuous but a discrete set of possible current values, it is thus improbable to get as close as possible to the singular value, as it is confirmed by the biological observation (Koch, 1999). There is one bio-physical condition which could contradict this fact: if there is a sustained sub-threshold oscillation, with a certain probability to cross the threshold, then there is no bound on the next spike-time. We assume here that, due to the leak, such oscillation is always damped. Reading of the literature does not provide a clear evaluation of this temporal bound, here we propose to assume that $\Delta T \simeq 100ms$, just to be sure not to underestimate it. This number is very useful at the simulation level, as detailed in the sequel.

A step further, in cortical layers, and especially in high (conductance) states (Destexhe et al., 2003), since neurons receive inputs at very high rates, it is experimentally observed that (almost ?) all neurons spike, with a rate not lower than about $1Hz$, thus with a maximal inter-spike interval T_{max} . Here we choose $T_{max} \simeq 2s$. This last property is however not related to the neuron itself but to its embedding in a given network environment. Thus, it has to be considered

²This is easy to illustrate considering a IIF model, where g and i are constant:

$$\begin{cases} C \frac{dV}{dt} + gV = i, \\ V(t_0) = V_0, V(t_1) = \theta \end{cases} \Rightarrow t_1 = t_0 + \frac{C}{g} \log \left(\frac{i - gV_0}{i - g\theta} \right) \text{ which requires } i > g\theta > gV_0.$$

In high state with $t_1 - t_0 < \Delta T$ the “equivalent current” must verify: $i > g(\theta - V_0 e^{-\Delta T g/C}) / (1 - e^{-\Delta T g/C})$. Since $C/g \simeq 1 \dots 10ms$, thus $e^{-\Delta T g/C} \ll 10^4$, it is sufficient to get $i > (1 + 10^{-4})g\theta$, i.e. a very small amount above $g\theta$. It is thus a reasonable numerical assumption to assume that ΔT is bounded in this case.

at another level.

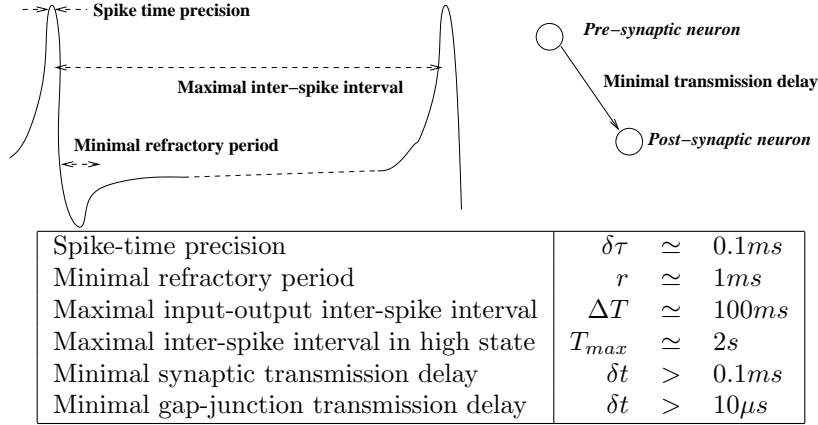


Figure 3: Summarizing the different characteristic times discussed in the text

2.2 Application: the maximal amount of information in a set of spikes

Summarizing, instantaneous firing is in fact a structural pathology of abstract Integrate and Fire models, whereas several useful characteristic times allow to better understand the behavior of spiking neurons and provide useful constraints for modeling and simulation as discussed in the sequel.

There is an important consequence: given a network of spiking neurons the information contained in the raster plot (i.e. in all spike times) is strictly bounded, since time is bounded and since two spike occurrences in a $\delta\tau$ window are not distinguishable, while spike time intervals are constrained between δt and T_{max} . At a very first glance, during one second, a unique neuron cannot code more information than when spiking or not-spiking as fast as possible with respect to its refractory period, thus $1/r$ bit of information per second. Furthermore, during a period of D second, with N neurons, it seems that we can code much more information, using for instance rank-coding (VanRullem

& Thorpe, 2001) i.e., taking the neurons spiking order into account. However, as discussed in (Viéville & Crahay, 2004) the spike-time precision imposes an important bound on the amount of information.

In order to evaluate this bound, let us consider the first spike time t_j^1 , this spike being fired by, say, the neuron of index j . A single neuron, (i) either fires not latter than $t_j^1 + \delta\tau$ thus at a time not distinguishable from t_j^1 by an observer, or (ii) fires at least $\delta\tau$ later. In order to be meaningful these first spikes must occur in distinct temporal boxes of width $\delta\tau$, the precise location of the box being fixed by the first time occurrence, as schematized in Figure 4. Since there is a refractory period of $r \gg \delta\tau$ the second and next spikes will never be mixed with their predecessor but are going to be subject to the same limitation.

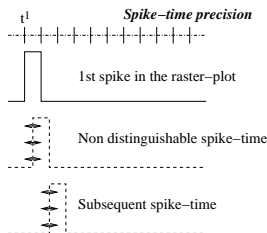


Figure 4: Evaluating the information in a set of spike times.

In average, this means that information coding can not be more than introducing for each neuron spikes every r seconds in a temporal histogram with $\delta\tau$ width temporal boxes. For each neuron, there are $D/\delta\tau$ choices for the first spike, less than $D/\delta\tau - 1$ for the second etc.. This means that for the D/r maximal number of spikes, they are less than $(D/\delta\tau)^{D/r}$ choices. Assuming that each neuron is independent, we obtain *an upper bound for the amount of information in a set of spike times*:

$$N \frac{D}{r} \log_2 \left(\frac{D}{\delta\tau} \right) \text{ bits during } D \text{ seconds}$$

Taking the numerical values into account we easily obtain (with $r = 1ms$ and assuming $\log_2(D) \gg \log_2(\delta\tau)$):

about $D \log_2(D)$ bits during D milliseconds for each neuron, providing $D \gg \delta\tau$

This means that rank-coding has not an “infinite precision” or “exponential precision” as sometimes suggested, but a much tighter bound. This bound is coherent with results presented in (Rieke, Warland, Steveninck, & Bialek, 1996) considering spike rate and using an information entropy measure. For instance, considering a timing precision of $0.1 - 1ms$ as derived here, the authors obtain an information rate bounded around $500bits/s$ for a neural receptor. The new point here, is that even if “each spike counts” due to time characteristics the bound has the same order of magnitude.

One particular case is fast-brain mechanisms where only “the first spike matters” (Thorpe & Fabre-Thorpe, 2001). In this case, considering rank-coding, the amount of information is not related to the permutations between neuron spikes, i.e. of order of $o(\log(N!)) = N \log(N)$ but simply proportional to N , in coherence to what is found in (Viéville & Crahay, 2004). Note that this not bad, but good news. For instance in statistical learning, this corresponds to a coding with large margins, thus as robust as support-vector machines likely explaining the surprisingly impressive performances of fast-brain categorization.

2.3 Reset to a constant value

Let us now discuss the second important aspect of the “fire” regime. The membrane potential is reset to a constant value. This has a dramatic effect. Changing the initial value of the membrane potential, one may expect some variability in the evolution. Now assume that there exists, for some neuron k , an interval of membrane potential values $V(0) \in [a, b]$, such that there is a time t where all trajectories coming from this interval are such that $V(t) \geq \theta$. Then, reset of the membrane potential maps the image of this interval to the point

V_{reset} . Then, all trajectories collapse on the same point and have obviously the same further evolution.

Though this effect can be considered as pathological as instantaneous firing, it has a great advantage. After reset, the membrane potential evolution does not depend on its past value. This induces an interesting property used in all the Integrate and Fire models that we know. *The dynamical evolution is essentially determined by the firing times of the neurons, instead of their membrane potential value.*

This is going to be deeply analyzed in a companion paper (Cessac & Viéville, 2007) after (Cessac, 2007) where this statement is going to be developed and made mathematically precise.

3 The “Integrate regime”

Position of the problem

The most general form for the membrane potential of a neuron (e.g. Hodgkin-Huxley) would include a non-linear conductance and an adaptive current depending on \mathbf{V} and some other dynamical variable. In the present section we are going to establish that *in Integrate and Fire models, this equation can be reduced to the specific form (1), where conductance and currents only depend on the firing times $\tilde{\omega}$ (up to time t), but not on \mathbf{V} , more precisely:*

$$C \frac{dV}{dt} + g(t, \tilde{\omega}) V = i(t, \tilde{\omega}). \quad (3)$$

This reduced equation is thus *linear* in V . Therefore, knowing the membrane potential at time t_0 and the list of spike times arrival one can obtain the

membrane potential at time t by:

$$V(t, \tilde{\omega}) = \nu(t_0, t, \tilde{\omega}) V(t_0) + \int_{t_0}^t \nu(s, t, \tilde{\omega}) i(s, \tilde{\omega})/C ds \quad (4)$$

with:

$$\nu(t, t', \tilde{\omega}) = e^{-\int_t^{t'} g(s, \tilde{\omega})/C ds} \quad (5)$$

If we choose for t_0 the last time spike so that $V(t_0) = V_{reset}$, V can now be written as a function of the spike times (thus of $\tilde{\omega}$) only.

This is an important characteristic which allows the derivation of theoretical results more easily than e.g. for the Hodgkin-Huxley's model. These are developed in (Cessac & Viéville, 2007). It also facilitates numerical simulations, as developed in the next sections. Note however, that the conductance and current may depend on the *whole past history* of the network.

We are going to obtain this derivation from usual realistic biological neuron models, so called bio-physical models (such as Hodgkin-Huxley models). More precisely we consider a specific form for the membrane potential equation of a neuron, “below the threshold”:

$$C \frac{dV}{dt} = -\frac{C}{\tau_L} (V - E_L) - I^{(adp)}(V, t, \tilde{\omega}) + I^{(ion)}(V, t, \tilde{\omega}) - I^{(syn)}(V, t) - I^{(gap)}(V, t) + I^{(ext)}(t) \quad (6)$$

thus with a leak characteristic time τ_L , a non-linear current $I^{(ion)}$ responsible for the spike generation (Brette & Gerstner, 2005), an adaptive current $I^{(adp)}$ responsible for the different firing regimes (phasic, tonic, bursting, ..) (Izhikevich, 2004), while synaptic currents $I^{(syn)}$, gap-junction currents $I^{(gap)}$ and external currents $I^{(ext)}$ are related to the neuron input.

Such modeling includes leaky integrate and fire models and the recently introduced “generalized Integrate and Fire models (gIF)”. That is, *conductance*

based models, where the action of neuron j on neuron k is manifested by a change in the *conductance* g (see equations (14),(16),(15) in appendix A).

In this section we discuss how the non-linear current $I^{(ion)}$ and the adaptive current $I^{(adp)}$ can be simplified in order the general equation (1) to (3). Other terms are already in the form of (3) and are reviewed in appendix A.

3.1 Adaptive regime $I^{(adp)}(V, t, \tilde{\omega}) \rightarrow I^{(adp)}(\tilde{\omega})$

The Fitzhugh-Nagumo reduction of the original Hodgkin-Huxley model (Hodgkin & Huxley, 1952) represents the average kinematics of the membrane channels by an adaptive current $I^{(adp)}(V, t, \tilde{\omega})$. Its dynamics is defined, between two spikes, by a second equation of the form:

$$\tau_w \frac{dI^{(adp)}}{dt} = g_w (V - E_L) - I^{(adp)} + \Delta_w \delta (V - V_\theta), \quad (7)$$

with a slow time-constant adaptation $\tau_w \simeq 144ms$, a sub-threshold equivalent conductance $g_w \simeq 4nS$ and a level $\Delta_w \simeq 0.008nA$ of spike-triggered adaptation current. It has been shown (Izhikevich, 2003) that when a model with a quadratic non-linear response is increased by this adaptation current, it can be tuned to reproduce qualitatively all major classes of neuronal in-vitro electrophysiologically defined regimes (e.g. bursting, using high reset values $E_L > E_A$; strong spike-frequency adaptation, using large spike-triggered adaptation current; sub-threshold oscillation using large sub-threshold equivalent conductance; overshoots in current pulse response using medium sub-threshold equivalent conductance, ..).

In order to further understand the action of this adaptive term, let us remark that that the time-constant adaptation is slow, and that when resetting the past dependence in the exact membrane potential value is removed. One may thus neglect the coupling with V for a short period of time. This writes:

$$\begin{aligned}
I^{(adp)}(V, t) &\simeq e^{-\frac{(t-t_0)}{\tau_w}} I^{(adp)}(t_0) + \frac{g_w}{\tau_w} \int_{t_0}^t e^{-\frac{(t-s)}{\tau_w}} (V(s) - E_L) ds + \Delta_w \#(t_0, t) \\
&\simeq \underbrace{e^{-\frac{(t-t_0)}{\tau_w}} I^{(adp)}(t_0) + g_w \left(1 - e^{-\frac{(t-t_0)}{\tau_w}}\right) (\bar{V} - E_L)}_{\text{slow variation}} + \underbrace{\Delta_w \#(t_0, t)}_{\text{spike-time dependent}}
\end{aligned}$$

where $\#(t_0, t)$ is the number of spikes in the $[t_0, t]$ interval while \bar{V} is the average value between t and t_0 .

As a consequence, this adaptive term is mainly governed by the spike-triggered adaptation current, the other part of the adaptive current being a standard leak³.

In other words *current adaptation is mainly due to spike occurrences*. We are going to verify this approximation in the experimental section. As a consequence, the adaptive current is no more directly a function of the membrane potential but function of the spikes. Hence we may consider the adaptive current as a function $I^{(adp)}(\tilde{\omega})$ of time and of the spikes produced before time t .

3.2 Non-linear response $I^{(ion)}(V, t, \tilde{\omega}) \rightarrow I^{(ion)}(\tilde{\omega})$

Here, $I^{(ion)}$ is a non-linear active (mainly Sodium and Potassium) current responsible for the spike generation. In models designed to simplify the complex structure of Hodgkin-Huxley equations, it is taken as quadratic or exponential, the latter form closer to observed biological data (Brette & Gerstner, 2005). It writes, for example:

$$I^{(ion)}(V) = \frac{C \delta_a}{\tau_L} e^{\frac{V-E_a}{\delta_a}} \text{ with } \left. \frac{dI^{(ion)}}{dV} \right|_{V=E_a} = \frac{C}{\tau_L}$$

³This last fact is also verified, by considering the linear part of the differential system of two equations (3,7) with $V, I^{(adp)}$ as unknowns, for an average value of the conductance $\bar{G}^+ \simeq 0.3 \dots 1.5 nS$ and $\bar{G}^- \simeq 0.6 \dots 2.5 nS$. It appears that the solutions are defined by two decreasing exponential profiles with $\tau_1 \simeq 16ms \ll \tau_2 \simeq 115ms$ time-constants, the former being very close to the membrane leak time-constant and the latter inducing very slow variations.

with $E_a \simeq -40mV$ being the threshold membrane state at which the slope of the I-V curve vanishes, while $\delta_a = 2mV$ is the slope factor which determines the sharpness of the threshold.

One key point here is that there is no need to define a precise threshold, since the neuron fires when the potential diverges to infinity. At the numerical level, a threshold is indeed to be defined but the value is not critical.

The second key point is that “below the threshold” the membrane potential is defined by a supra-linear kinematics ($\frac{dV}{dt}$ is higher than what is predicted by a linear conductance equation). However, it is known that no explicit solution for the membrane potential is available in the general case, as soon as the conductance or current are not constant.

A recent contribution (Touboul, 2007) re-analyzes such non-linear currents and shows that we obtain the correct dynamics providing that the profile is mainly non-negative and strictly convex, not necessarily a quadratic or exponential function⁴. It is thus interesting to work not with “one” form of ionic current but a family. A step further, instead of choosing a stationary current $I^{(ion)}(V)$ depending only on the membrane potential, one could *introduce a non-stationary current* $I^{(ion)}(V, t, \tilde{\omega})$ *depending also on the previous spike times*, which is plausible considering the membrane dynamic properties.

In order to proceed let us write $i(V, t, \tilde{\omega}) = i'(t, \tilde{\omega}) + I^{(ion)}(V, t, \tilde{\omega})$ thus separate the $I^{(ion)}$ from all other currents written $i'(t, \tilde{\omega})$. Let us consider the last spike time t_0 of this neuron and let us write \tilde{V} the solution of the linear differential equation “without” the ionic current $I^{(ion)}$:

$$C \frac{d\tilde{V}}{dt} + g(t, \tilde{\omega}) \tilde{V} = i'(t, \tilde{\omega})$$

with $V(t_0) = V_{reset}$, as obtained in (4). Define now $\hat{V} = V - \tilde{V}$, with $\hat{V}(t_0) = 0$, V being the solution of (1). Then (3) yields:

⁴In addition, the author proposes an original form of the ionic current, with an important sub-threshold characteristic not present in previous models (Izhikevich, 2003; Brette & Gerstner, 2005).

$$C \frac{d\hat{V}}{dt} + g(t, \tilde{\omega}) \hat{V} = I^{(ion)}(\hat{V} + \tilde{V}(t, \tilde{\omega}), t, \tilde{\omega})$$

as easily obtained by superposition of the linear parts of the equation.

Let $h(t, \tilde{\omega})$ be any regular function and $f(V)$ any bijective regular function with $f(\hat{V}) \neq 0$. These two functions allow to model a whole family of ionic currents:

$$I^{(ion)}(\hat{V} + \tilde{V}(t, \tilde{\omega}), t, \tilde{\omega}) = g(t, \tilde{\omega}) \hat{V} + \frac{h(t, \tilde{\omega})}{f(\hat{V})}. \quad (8)$$

The choice of H and f is simply related to specific properties: The reader can easily verify that it allows to obtain a closed form:

$$\hat{V}(t, \tilde{\omega}) = F^{-1} \left(\int_{t_0}^t h(s, \tilde{\omega}) ds \right) \text{ with } F' = f \text{ and } F(0) = 0. \quad (9)$$

so that \hat{V} is now a function of $\tilde{\omega}, t$ with $\hat{V}(t_0, \tilde{\omega}) = 0$, and so is $I^{(ion)}(\hat{V}(t, \tilde{\omega}) + \tilde{V}(t, \tilde{\omega}), t, \tilde{\omega})$, removing the direct dependence on V . In other words, it now depends only on t and on the spike times (thus on $\tilde{\omega}$) and not anymore on the membrane potential explicitly. Clearly, this only applies to neurons which have fired at least once during the period of observation. Otherwise, we assume that its initial condition was also V_{reset} . Let us instantiate this formal derivation by two illustrative examples:

Exponential ionic current with adaptive threshold Let us choose:

$$\begin{aligned} I^{(ion)}(V, t, \tilde{\omega}) &= \frac{C \delta_a}{\tau_L} e^{\frac{V(t) - E_a(t, \tilde{\omega})}{\delta_a}} \\ E_a(t, \tilde{\omega}) &= \tilde{V}(t, \tilde{\omega}) - \delta_a \ln \left(\frac{g(t, \tilde{\omega})}{\bar{g}} \right) \end{aligned} \quad (10)$$

for any $\bar{g} > 0$ which allows to control the threshold for different conductance.

Here $h = g$ and $f(v) = (k e^{\frac{v}{\delta_a}} - v)^{-1}$ for some k . This corresponds to the voltage profile show in Figure 5.

In this case the threshold is no more fixed, but adaptive with respect to

$g(t)$: the higher the conductance, the higher the threshold (via the \bar{V}). This is coherent with what has been observed experimentally (Azouz & Gray, 2000; Wilson, Weyrick, N. E. Hallworth, & Bevan, 2004), since the higher the conductance, the higher the frame rate increases with the spiking threshold.

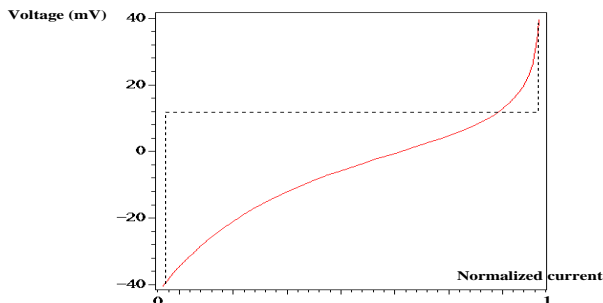


Figure 5: Showing the F^{-1} curve for an exponential ionic current with adaptive threshold (plain curve). The abscissa scale has been normalized. In this case the I-V curve slope vanishes around $20mV$ as schematized by the dashed lines.

Two stages delayed non-linear current When targeting fast implementation, instead of considering the straightforward but rather heavy previous formulas, another qualitative interpretation of the previous derivation could be given.

Qualitatively, $I^{(ion)}(V, t, \tilde{\omega})$ is most of the time negligible with respect to other currents but then targets the next spiking time by a huge jump in current so that the I-V curve slope vanishes, while all other components becomes negligible. In other words, it acts as a two-stage variable first silent then targeting the spike. Now we have shown how it is reasonable to assume that this current can be computed “in advance” i.e. knowing the previous spike times $\tilde{\omega}$.

Putting these two facts together, this ionic current can be implemented as a “programmed timer”, enforcing the neuron to spike after a given delay

which value is adjusted at each time arrival. The delay may be infinite.

This is going to be illustrated in the next section.

Conclusion

We have been able to reduce (6) to (3):

1. Considering that the adaptive current influence is mainly related to spike occurrence and approximating the continuous part of this influence by some leak,
2. Representing the non-linear part of the membrane potential by a current function of spike times only.

At the network level, though equation (3) looks like a factorization of the dynamics into N independent differential equations, it is not. Indeed, the evolution of V depends (via $\tilde{\omega}$) on the evolution of *all* neurons. This is going to be discussed in another contribution (Cessac & Viéville, 2007).

Let us now switch from theory to applications and illustrate how these general considerations have important consequences for the simulation of models of biological neurons. In the sequel, additional choices are to be made, but always driven by the previous discussion.

4 Application: one neuron spiking regime

The numerical and symbolic analysis of the neuron bio-physical model in section 3 allow us to claim that (6) can be reduced in the form of (3). This has to be verified numerically. This is the goal of this section.

In order to experiment this point we consider a very simple model whose

evolution equation at time t for the membrane potential v is:

$$\begin{aligned}
& \text{if } (t = 0) \\
& \qquad v = 0; u = 0; t_0 = 0; \\
& \text{else if } (v \geq \theta) \\
& \qquad v = V_{reset}; u = u + k; t_0 = t; \qquad (11) \\
& \text{else} \\
& \qquad \dot{v} = -g (v - E) - u + i; \\
& \qquad \text{if } (t > t_0 + d) v = 0
\end{aligned}$$

where u is the adaptive current (entirely defined by equation (11)), t_0 the last spiking time, d the non-linear current delay, θ a fixed threshold and V_{reset} a fixed reset potential. The differential equation is simulated using an Euler interpolation as in (Izhikevich, 2003; Touboul, 2007) to compare our result to what has been obtained by the other authors. The input current i is either a step or a ramp as detailed in Figures 7 and 1.

However, obviously, there is a closed form solution for the next spiking time allowing a direct implementation in event-based simulators.

Four parameters, the constant leak conductance g , the reversal potential E , the adaptation current k step and the (eventually infinite) non-linear current delay d allows to fix the firing regime. These parameters are to be recalculated after the occurrence of each internal or external spike. In the present context, it was sufficient to use constant value except for one regime, as made explicit in Figure 1.

Following section 3, the non-linear response is not related to the membrane potential, whereas an additional “internal” current is introduced. In the present case, we use the two-stages ionic current whose action is to reset the membrane current after a certain delay. We made this choice because it was the simplest.

Several alternatives are indeed possible. The present choice leads to a very fast implementation.

Experimental results using a tiny simulator shown in Figure 6 are given in Figure 7 for the parameters listed in Figure 1. With such a simple model, the membrane potential itself is indeed unrealistic. These results correspond to all well-defined regimes proposed in (Izhikevich, 2003). See (Touboul, 2007) for a discussion about other regimes. However, the raster plot corresponds to what is expected, which is the goal here. The parameter adjustment is very easy to obtain as soon as the bifurcation diagram is known, see (Touboul, 2007) for a discussion about this point.

These results corroborate what has been proposed for the bio-physical model reduction. They allow to place a new point on the performance/efficiency plane proposed by (Izhikevich, 2004) at a very challenging place. No mistake, however, this is a “provocative” result, because if we experimentally simulate all well-defined regimes of a neuron in terms of spike times, all the story is not in this over-simple simulation proposed in this section. The membrane potential calculated here is totally different with respect to what is observed in the biology, synaptic input are not taken into account, etc.. as it is the case in (Brette & Gerstner, 2005; Touboul, 2007). It is a perspective of this work, to revisit section 3 and propose a more realistic neuron simulation.

Here, we simply show that several regimes can be simulated with an equation of the form of (3), simpler than (1).

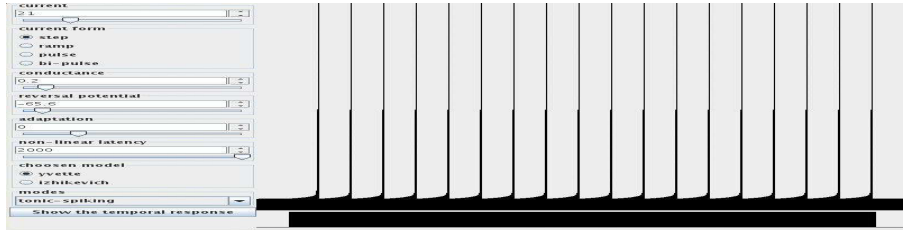


Figure 6: A tiny simulator allows to reproduce the neuronal qualitative behaviors using the present bio-physical model reduction. Here a phasic-spiking mode is shown. The upper trace shows the action potentials, the lower trace the input current.

spiking mode	leak conductance g	reverse potential E	adaptation step k	non-linear delay d	input magnitude $i(t)$	input form
phasic-spiking	0.04	0	30	$+\infty$	0.5	step
tonic-bursting	0.18	1.6	14.6	60	15	step
phasic-bursting	0.06	11	11.2	$+\infty$	0.5	step
mixed-mode	0.01	0	K	150	10	step
resonator	0.04	-27	0	$+\infty$	38	bi-pulse
bistability	0.88	80	1.8	$+\infty$	65	pulse
positive CFR	0.01	0	0	$+\infty$	30	ramp
negative CFR	0.52	80	4	$+\infty$	30	ramp
constant CFR	0.52	0	4	100	30	ramp

Table 1: Examples of parameters used to generate the spiking modes shown in Figure 6 and Figure 7. The mixed mode is simulated by a variable adaptation step $k = \{-20, 20\}$.

5 Application: next spike time calculation

In order to calculate the next spike time t_θ , we have to solve, the previous spike-times $\tilde{\omega}$ being fixed, from (4) and (5):

$$\begin{aligned}
 V(t_\theta) &= \theta \\
 V(t) &= \nu(t_0, t) V(t_0) + \int_{t_0}^t \nu(s, t) i(s) / C ds \\
 &\quad \text{where } \log(\nu(t, t')) = - \int_t^{t'} g(s) / C ds
 \end{aligned}$$

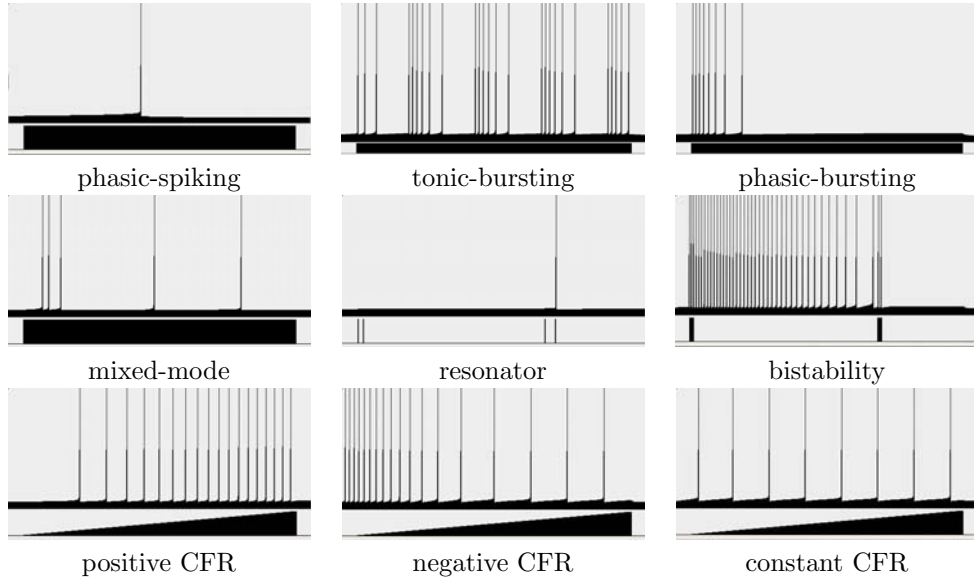


Figure 7: Typical results showing the versatility of the reduced model for spiking, bursting and other modes, including and different current-frequency-responses (CFR). For each mode, the upper trace shows the action potentials, the lower trace the input current. These results include the excitatory mode of type I where the spike frequency can be as small as possible in a $1 - 10^3 Hz$ range and of type II where the spike frequency remains bounded.

In order to proceed, let us consider:

$$V(t) < V(t_0) + D(t_0)(t - t_0), t \geq t_0 \quad (12)$$

for some $D(t_0)$ derived in appendix B and where $V(t_0) < \theta$ can be computed numerically from (4) thanks to section 3. This method is only available for the conductance model proposed here (where the ionic and adaptive currents being redefined in function of the spike-times only). This allows us to derive a lower bound of the next-spike time:

$$\begin{cases} t_\theta = +\infty & , D(t_0) \leq 0 \\ t_\theta > t_0 + \frac{\theta - V(t_0)}{D(t_0)} & , D(t_0) > 0. \end{cases} \quad (13)$$

Given a lower-bound, we introduce a new efficient strategy in event-based simulations. When the next spike time is required, it is allowed not to provide the exact value but only a lower-bound. When the simulator time reaches this lower-bound time, the next spike time lower-bound approximation is required again, providing a better approximation. This corresponds, at a very general level, to an iterative estimation of the spike-time when the exact value is not available. The convergence of the series:

$$t_{n+1} = t_n + \frac{\theta - V(t_n)}{D(t_n)}$$

is guaranteed by the fact it is by construction a strictly increasing series bounded by t_θ . In practice, the convergence is obtained when $|t_{n+1} - t_n| < \delta\tau$.

It provides a challenging alternative solution to the event-based simulation of gIF models (Rudolph & Destexhe, 2006; Rochel & Martinez, 2003; Brette et al., 2007).

Regarding the computational complexity, the analysis of the equations made in appendix B demonstrates that the number of operations can be bound to $O(T) + O(S)$ where T is the simulation time and S the number of spike input to one neuron and not to $O(TS)$ as a naive implementation would have lead to. This is due to the fact that exponential and alpha profiles are easy to calculate with recursive equations (Anderson & Moore, 1979).

6 Application: event-based simulation of spiking networks

Let us derive another important consequence of the previous analysis: the capability to dramatically improve event-based simulation of spiking networks.

In event-based simulation, the next spike-time is computed explicitly for each neuron and the network simulation runs on the following principle:

- At a given time the neuron firing next (i.e. which next spike-time is the smallest) is selected.
- The simulation clock is incremented. The firing neuron and all connected neurons update their next spike-time, taking this event into account.

Both steps are repeated while neurons fire. This provides (in principle) an unbiased solution (Rochel & Martinez, 2003) for IF neurons, or any other model of neurons, whenever the next spike-time can be computed with an *analytic form*. Such an event-based simulation is not only very fast, allowing large assemblies of neurons to be simulated, dealing with the difficult problem of high temporal precision requirement, but it also avoids several implementation problems (e.g. synchronous versus asynchronous update, temporal approximations) occurring with analog network simulations or clock-based simulators. See (Brette et al., 2007) for a recent comparative review of this method with respect to clock-based methods.

There is however a key problem here: spiking-time must be sorted at each step. The sorted sequence of spike-times is indeed to be implemented in an efficient data structure such as a *B-tree* (e.g. (Mattia & Giudice, 2000)) or an equivalent structure in order to provide an optimal $o(\log_2(N))$ insert time and also as a double-chained list in order to provide a $o(1)$ access time to the first item of the sequence. Can we avoid this $o(\log_2(N))$ complexity? Thanks to what has been developed in section 2 there is a positive answer based on two remarks:

Upper bound At a given step, the next spike-times are bounded by ΔT (i.e. the neuron⁵ will either fire before ΔT in the future or never fire), unless another spike occurs, while we assume the input current to be constant between spikes, as discussed in appendix A.

⁵Since ΔT may depend on the neuron, the maximal value is considered.

Lower bound Given a spike starting at time t , from the previous discussion, spikes induced by this spike will always occur at least δt later, while other spikes starting before $t + \delta\tau$ can be considered as synchronous. When a neuron firing in this time window is selected, due to the transmission delay, it always induce changes which are to be taken into account after δt in the future. This means that it has no influence over the other spike-times within the same δ_ϵ window.

This means that there is a time window $\delta_\epsilon < \min(\delta t, \delta\tau)$ where we can manage the spikes in any order, without changing the subsequent simulation.

This also avoids any problem of causality and any complex management of synchronicity as in event-based simulators where these time characteristics are not made explicit (Rochel & Martinez, 2003).

Here we choose⁶ $\delta_\epsilon = 1\mu s$.

This allows us to propose to simply sort spike-time estimations in an histogram of size $\Delta T/\delta_\epsilon = 10^5$ each box corresponding to a δ_ϵ step. At the implementation level this histogram corresponds to a ring-buffer with contains all spike-time predictions for the next ΔT , while the past information is removed.

Each box contains a list of all neurons with spike-times within a given δ_ϵ window, although *spike-times are themselves not sampled, but calculated at the machine precision*.

With this algorithmic mechanism the complexity of spike-time insertion is not $o(\log_2(N))$ anymore but simply $o(1)$. This reduction of complexity is so important that the simulation kernel reduces to about 100 lines of C/C++ altogether.

⁶Here, we choose such a very small bound ($10\mu s$ would have been very likely sufficient) in order to be sure to avoid any “clock-based” spurious effect (see (Rudolph & Destexhe, 2007) for a discussion). It also allows to simulate biologically implausible models in order to compare their properties with biologically plausible ones.

If the upper-bound assumption or the lower-bound assumptions is violated the kernel detects a spurious indexing of the histogram ring buffer and throws a fatal exception. This never happened during the tests, validating the method.

All other aspects of the simulator are directly derived from the MVASPIKE simulator (Rochel & Martinez, 2003), actually the most powerful event-based simulator known in the field. The present method is a constrained instance of this more general mechanism, taking the assumptions discussed in section 2 into account and optimized accordingly.

In order to validate this idea, we have reproduced the benchmark proposed in (Brette et al., 2007) which is dedicated to event-based simulation: it consists of 4000 IF neurons, which 80/20% of excitatory/inhibitory neurons, connected randomly using a connection probability of $1/32 \simeq 3\%$. So called “voltage-jump” synaptic interactions are used: the membrane potential is abruptly increased/decreased by a value of $0.25/2.25mV$ for each excitatory/inhibitory event. Here, we also introduce a synaptic delay of $2/4ms$ respectively and an absolute refractory period of $1ms$, both delays being corrupted by an additive random noise of $10\mu s$ of magnitude. We also have increased the network size and decreased the connection probability to study the related performances. See (Brette et al., 2007) for further details.

In term of performances, on a standard portable computer (Pentium M 750 1.86 GHz, 512Mo of memory) we process about 10^7 spike-time updates / second, given the network size and connectivity, although the code is slightly slowed down by check-points to validate the method. Performances details are reported in Figure 2. It confirms that the algorithmic complexity only marginally depends on the network size, while it is mainly function of the number of synapses, as also confirmed when profiling the code. We also notice a tiny overhead when iterating on empty boxes in the histogram, mainly visible when the number of spike is

small. This overhead is constant for a given simulation time. In a nutshell this methods allows about 10^{6-7} updates (spike-time insertion/deletion) pro second. The lack of proportionality is due to the introduction of some optimization in the evaluation of spike-times, which are not updated if unchanged. We also have tested the performances using randomly spiking neurons and several other set of parameters have been used, not reported in Figure 2, since they simply corroborate the explained results.

Network size	Number of synapses	CPU time	Simulation time	Number of 10^6 updates/second	Proportion of active neurons	Upper bound	Lower bound
2^{12}	$\simeq 2^{19}$	0.96s	0.10s	14	92%	0.1s	1 μ s
2^{13}	$\simeq 2^{21}$	2.2s	0.11s	12	96%	0.1s	1 μ s
2^{14}	$\simeq 2^{23}$	7.1s	0.11s	8	96%	0.1s	1 μ s
2^{15}	$\simeq 2^{25}$	18s	0.11s	6	98%	0.1s	1 μ s
2^{16}	$\simeq 2^{27}$	48s	0.10s	4	99%	0.1s	1 μ s
2^{16}	$\simeq 2^{26}$	22s	0.10s	5	98%	0.1s	1 μ s
2^{16}	$\simeq 2^{25}$	11s	0.10s	5	97%	0.1s	1 μ s
2^{16}	$\simeq 2^{24}$	5.5s	0.10s	5	92%	0.1s	1 μ s
2^{13}	$\simeq 2^{21}$	2.4s	0.11s	11	96%	1s	1 μ s
2^{13}	$\simeq 2^{21}$	2.1s	0.12s	12	96%	1s	10 μ s
2^{13}	$\simeq 2^{21}$	2.1s	0.12s	12	96%	0.1s	10 μ s

Table 2: Simulation performances, for 10^5 spike firings, varying the network size and number of synapses (via the connection probability). The performances measured by the CPU time is mainly function of the number of synapses, not the number of neurons. The spike-time structure upper and lower bounds have a marginal influence on the performances, see text for details.

A step further, we also made profit of the equations derived in section 5 to reproduced benchmarks close to what is proposed in (Brette et al., 2007) with current-based interactions (CUBA model) and another one with conductance-based interactions (COBA model). In our context, current based interactions correspond to gap junctions, while conductance-based interactions correspond to synaptic junctions. However, in the COBA original models, when a spike occurs, the synaptic conductance is instantaneously incremented by a quantum value (6 nS and 67 nS for excitatory and inhibitory synapses, respectively) and decayed exponentially with a time constant of 5 ms and 10 ms for excitation

and inhibition, respectively, whereas here an “alpha” profile is used. In CUBA models, the conductance quanta is of 0.27 nS and 4.5 nS for excitatory and inhibitory synapses, respectively. Despite the use of “exponential” profiles instead of “alpha” profiles and a few numerical approximations at the implementation level, we have obtained similar results as illustrated in Figure 8, as reported in (Brette et al., 2007).

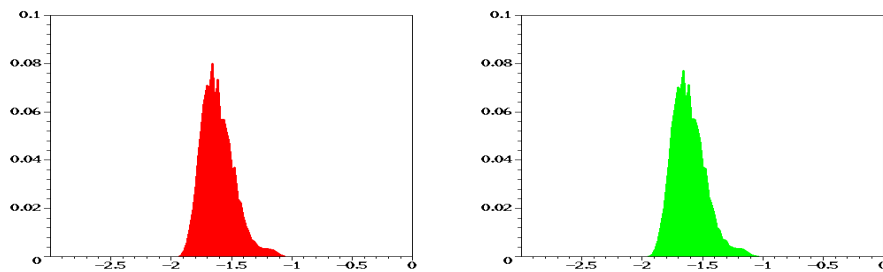


Figure 8: Inter-spike interval histogram for excitatory neurons (in red) and inhibitory neurons (in green). The abscissa is the decimal log of the interval and the ordinate the inter-spike observed probability. Results qualitatively corresponds to the expected result for this kind of benchmark, with a distribution from 10ms to more than 100ms with a peak around 50ms similar for both excitatory and inhibitory neurons. Here, there are inter-spike intervals higher than 100ms up to 1s but only with a residual quantity.

7 Conclusion

Revisiting the characteristic times of the gIF firing and proposing two new approximations for the reduction of bio-physical model of a punctual neurons allow us to reconsider some of the well accepted aspects of biological neuron simulation. At the theoretical level it eliminates spurious properties of the biological model and has important consequences. One is the amount of information coded in a set of spike time. Another is the detailed analysis of the dynamics at the network level detailed in the companion paper (Cessac & Veville, 2007). At the

numerical level it leads to several interesting applications: simulation of several spiking regimes with a modified IIF neuron, calculation of the next spike in complex gIF neurons and efficient event-based simulation of spiking networks.

In a nutshell, an intermediate solution between rather complex gIF models in the form of (1) and IIF is proposed in the form of (3). It seems to have a power of representation close to complex gIF models but with computational properties close to IIF. As it, it is thus a challenging tool, based on another compromise in terms of approximations.

At this point there is however a delicate question related to the mathematical developments based on such approximations. Because of the initial condition sensitivity due to the threshold, any approximation may provoke, on a finite time scale, some large difference between the true trajectory and the simulated trajectory. One may expect this type of discrepancy when some neurons are close to the threshold (Destexhe et al., 2003). This problem is extensively discussed in (Cessac, 2007) where it appears that suitable numerical experiments using a pertinent indicator of the network “effective entropy” allows to verify if such approximations are qualitatively coherent. This the next step of this work, detailed in (Cessac & Viéville, 2007).

A Bio-physical model reduction

For this paper to be self-contained and because it is used in the simulation, let us review the components of equation (6) not discussed in section 3. We follow (Izhikevich, 2004; Brette & Gerstner, 2005; Rudolph & Destexhe, 2006) in this section.

Voltage range

The membrane potential, outside spiking events, verifies:

$$V(t) \in [V_{\text{reset}}, V_{\theta}]$$

with typically $V_{\text{reset}} \simeq E_L \simeq -80\text{mV}$ and a threshold value $V_{\theta} \simeq -50\text{mV} \pm 10\text{mV}$ as illustrated in Figure 9.

The reset value is typically fixed, whereas the firing threshold is inversely related to the rate of rise of the action potential upstroke (Azouz & Gray, 2000). This adaptive threshold mechanism is discussed in details in section 3, since introduced via some diverging non-linear ionic current $I^{(ion)}$ as in (Izhikevich, 2004; Brette & Gerstner, 2005).

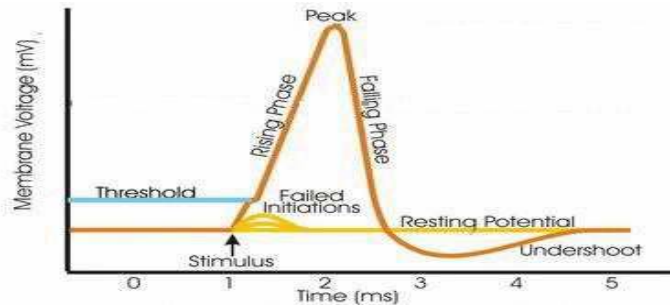


Figure 9: A schematic view of the membrane potential. Between two action potentials, i.e. spikes, the potential is between its “reset” and “threshold” value. When the threshold is reached an action potential of about 1-2 ms is issued and followed by refractory period of 2-4 ms (more precisely, an absolute refractory period of 1-2 ms without any possibility of another spike occurrence followed by a relative refraction to other firing). Voltage peaks are at about 40mV and voltage undershoots about -90mV . The threshold is in fact not sharply defined, as discussed in the text.

Membrane passive properties

The membrane leak time constant $\tau_L \simeq 20\text{ms}$ ($\tau_L = C_L/G_L$ with $C_L \simeq 1\mu\text{Fcm}^{-2}$ the membrane capacity and $G_L \simeq 0.0452\text{mScm}^{-2}$ the membrane passive conductance) is defined for a reversal potential $E_L \simeq -80\text{mV}$, while

$C = SC_L$ is the membrane capacity (with a membrane area $S \simeq 38.013 \mu m^2$). Here $C \simeq 300 pF$ and $G_L \simeq 20 nS$. We shall assume that all neurons have the same characteristic constants.

Input current $I^{(ext)}$

Direct input (or external) current $I^{(ext)}$ is often related to electro-physiological clamps. At another level of representation, the average activity of the neuron can be modeled by a constant or random input current. Let us maintain the possibility to introduce such an additive term here, since it does not change the nature of the problem.

In the sequel we further assume that this current is constant between two spikes, e.g. because its temporal variation is small enough to be neglected.

Synaptic current $I^{(gap)}$

In conductance based model the occurrence of a post synaptic potential on synapse j results in a change of the *conductance* of neuron k . Consequently, we write synaptic current received by neuron k in the form:

$$I^{(syn)}(V, t) = \sum_j G_j^+(t, \tilde{\omega}) [V(t) - E_+] + \sum_j G_j^-(t, \tilde{\omega}) [V(t) - E_-], \quad (14)$$

where conductance are positive $G_j^\pm(t, \tilde{\omega}) \geq 0$ and depends on previous spike-times $\tilde{\omega}$. The indexes $+$, $-$ correspond to the cumulative effect of excitatory (typically, in real neural networks 10^4) and inhibitory (typically $2 \cdot 10^3$) synapses. The corresponding reversal potential are $E_+ \simeq 0 mV$ and $E_- \simeq -75 mV$, usually related to AMPA and GABA receptors. The conductance typically evolves in time. Indeed, for one spike, the conductance time-course is usually modeled by so called “alpha” profiles, as detailed in Figure 10:

$$G_j^\pm(t) = \bar{G}_j^\pm \alpha^\pm(t - \delta t_j),$$

where $\delta t_j = 1..10ms$ is the inter-neuron delay, with:

$$\alpha^\pm(t) = H(t) \frac{t}{\tau^\pm} e^{-\frac{t}{\tau^\pm}} \quad (15)$$

where H is the Heaviside function (related to causality). Typically, in biological neurons, in average: $\bar{G}_j^+ \simeq 0.66 nS, \tau^+ \simeq 2 ms$ and $\bar{G}_j^- \simeq 0.63 nS, \tau^- \simeq 10 ms$ for excitatory and inhibitory synapses respectively. We shall assume that the characteristic times τ^\pm do not depend on the synapse. On the contrary, the coefficients \bar{G}_j^\pm give a measure of the synaptic strength (unit of charge) and vary from one synapse to another and are also subject to adaptation.

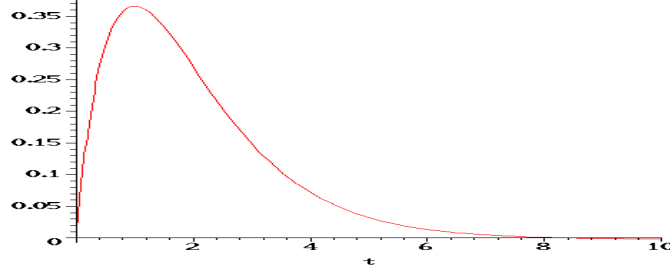


Figure 10: The alpha profile $\alpha(t) = H(t) \frac{t}{\tau} e^{-\frac{t}{\tau}}$ plotted here for $\tau = 1$. It is maximal at $t = \tau$ with $\alpha(\tau) = 1/e$, the slope at the origin is $1/\tau$ and its integral value $\int_0^{+\infty} \alpha(s) ds = \tau$ since $(\int \alpha)(t) = (\tau - t) e^{-\frac{t}{\tau}} + k$. This profile is concave for $t \in]0, 2\tau[$ and convex for $t \in]2\tau, +\infty[$, while $\alpha(2\tau) = 2/e^2$ at the inflexion point.

The choice of such a post-synaptic potential profile is twofold: it is relatively close to what is observed in biological measurements and it is a continuous potential, whereas using a discontinuous curve (e.g. using “exponential” profiles) is also of common use but introduces some artificial discontinuities with possible model-induced spurious effects.

Comparison with two-state kinetics profiles. A step ahead, two-state kinetics profiles of the form:

$$\alpha'(t) = H(t) \frac{1}{\kappa-1} (e^{-\frac{t}{\tau}} - e^{-\kappa \frac{t}{\tau}})$$

are another biologically plausible representation of the post-synaptic potential time-course, with an additional parameter $\kappa > 1$, as illustrated in Figure 11 and discussed in (Rudolph & Destexhe, 2006) or (Gerstner & Kistler, 2002). We mention this alternative because it is closer to what is obtained from a bio-chemical model of a synapse. However, it is not clear whether the introduction of this additional degree of freedom is significant here (for instance the slope at the origin and the profile maximal value can be adjusted independently with two-state kinetics profiles, which is not really useful in this context). Furthermore, the two-state kinetics profile is related to the alpha-profile by the relation:

$$\lim_{\kappa \rightarrow 1} \alpha'(t) = \alpha(t)$$

It is anyway straightforward to apply this alternative to our developments. Here, for clarity, we only explicit alpha-profiles in the sequel because this is the most commonly used profile in the literature.

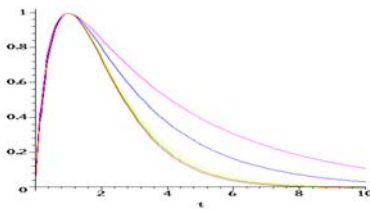


Figure 11: The two states kinetic profile is plotted with a normalized magnitude for the same $\tau = 1$ but different $\kappa = 1.1, 1.5, 2, 5, 10$ showing the effect of this additional degree of freedom. It is maximal at $t_{\bullet} = \tau \ln(\kappa)/(\kappa - 1)$ the slope at the origin is $1/\tau$ and its integral value τ/κ . The profile is concave for $t \in]0, 2t_{\bullet}[$ and convex for $t \in]2t_{\bullet}, +\infty[$.

Given this alpha-profile, upon a spike arrival on synapse of index j , at time t_j^n , the conductance vary according to an additive rule:

$$G_j^{\pm}(t) \rightarrow G_j^{\pm}(t) + \bar{G}_j^{\pm} \alpha^{\pm}(t - t_j^n - \delta_j).$$

Here we consider that $G_j^{\pm}(0) = 0$, since in the absence of spike, the synaptic conductance vanishes (Koch, 1999).

Thus, the update of the membrane potential and synaptic conductance needs

the knowledge of spikes arrival $t_j^1, \dots, t_j^n \dots$. Hence, the synaptic conductance G_j^\pm at time t are function of the list of firing times occurring on j within time interval $[0, t]$. This sequence can be encoded in the fire-time set $\tilde{\omega}$ where G_j becomes a function of $\tilde{\omega}$:

$$G_j^\pm(t, \tilde{\omega}) = \bar{G}_j^\pm \sum_{n=1}^{M_j(t, \tilde{\omega})} \alpha^\pm(t - t_j^n - \delta_j) \quad (16)$$

where \bar{G}_j^\pm is the synaptic strength and $M_j(t, \tilde{\omega})$ is the number of spikes that has arrived between 0 and t on synapse j . Note that we assume in (16) that there are finitely many spikes arising within a finite time interval.

The proposed model affords straightforward extensions involving synaptic plasticity (e.g. STDP, adjusting the synaptic strength), that will be discussed in a separate paper.

Gap junctions $I^{(gap)}$

It has been recently shown, that many local inter-neuronal connections in the cortex are realized though electrical gap junctions (Galarreta & Hestrin, 2001), this being predominant between cells of the same sub-population (Amitai et al., 2002). At a functional level they seem to have an important influence on the synchronicity between the neuron spikes (Lewis & Rinzel, 2003). Such junctions are also important in the retina (Wohrer, Kornprobst, & Viéville, 2006).

The electrotonic effect of both the sub-threshold and supra-threshold portion of the membrane potential $V_j(t)$ of the pre-junction neuron of index j seems an important component of the electrical coupling. Considering the previous discussion, this writes (see e.g. (Wohrer et al., 2006) or (Lewis & Rinzel, 2003) for a discussion about such resistive connections between neuron):

$$I^{(gap)}(V, t) = \sum_j G_j^* \left[[V_j(t) - V(t)] + E_\bullet \sum_{n=1}^{M_j(t, \tilde{\omega})} \beta(t - t_j^n - \delta_j^\bullet) \right]$$

where G_j^* is the electrical coupling conductance, the term $V_j(t) - V(t)$ accounts

for the sub-threshold electrical influence and $E_{\bullet}\beta()$ is the spike supra-threshold voltage influence. The neuronal media also induces some electrical diffusion not made explicit in the equation.

Here \bullet indexes the gap junction as $+$ and $-$ indexes excitatory and inhibitory synaptic junctions.

In order to propose a choice coherent with respect to the previous synaptic junctions, we consider the following profile:

$$\beta(t) = e\alpha^{\bullet}(t)$$

for a time constant of $\tau_{\bullet} \simeq 1ms$ which is the spike raise time, while $E_{\bullet} \simeq 80mV$ corresponds to the usual spike voltage magnitude of the spiking threshold.

This profile corresponds to the supra-threshold voltage which is slightly filtered by the biological media. It is not clear whether this choice is optimal, but it seems reasonable with respect to biological data (Galarreta & Hestrin, 2001; Lewis & Rinzel, 2003) and it allows to take gap junction into account with a minimal increase of complexity.

A step further, we propose to neglect the sub-threshold term for three main reasons. On one hand, obviously the supra-threshold mechanism is higher than the sub-threshold mechanism, especially in the temporal domain since the former has a large dynamics with respect to the latter which is also smoothed by the media diffusion. On the other hand, this electrical influence remains local (quadratic decrease with the distance) and is predominant between cells of the same sub-population which are either synchronized or have a similar behavior, as a consequence $|V_j(t) - V(t)|$ remains small for cells with non-negligible electrical coupling. Furthermore, a careful analysis of such electrical coupling (Lewis & Rinzel, 2003) clearly shows that the sub-threshold part of the contribution has not an antagonist effect on the neuron synchrony, i.e., it could be omitted without changing qualitatively the gap junction function.

Finally, since $\delta_j^\bullet \ll 1ms$ we neglect the gap junction delay, regarding the integrate regime (although we take it into account for the fire regime, as discussed previously).

We finally obtain:

$$I^{(gap)}(V, t) = E_\bullet \sum_j G_j^\bullet(t, \tilde{\omega}) \text{ with } G^\bullet(t, \tilde{\omega}) = G_j^\bullet \sum_{n=1}^{M_j(t, \tilde{\omega})} \alpha^\bullet(t - t_j^n) \quad (17)$$

with $G_j^\bullet = e G_j^*$ obtaining a form similar to (16).

B Next spike time lower bound

Linear lower-bound calculation

Let us thus derive a quantity $D(t_0)$ as proposed in (12), from (6), (14), (17), (16), (15) defined in section 3 and appendix A. On one hand:

$$\begin{aligned} g(t, \tilde{\omega})/C &= \frac{1}{\tau_L} + \sum_{j=1}^N G_j^+(t, \tilde{\omega})/C + \sum_{j=1}^N G_j^-(t, \tilde{\omega})/C \\ &= \frac{1}{\tau_L} + \sum_{\diamond=\{+, -\}} \sum_{j=1}^N \bar{G}_j^\diamond/C \sum_{n=1}^{M_j(t, \tilde{\omega})} \alpha^\diamond(t - \delta t_j - t_j^n) \quad (18) \\ &= \frac{1}{\tau_L} + \sum_l g_l \alpha^l(t - t_l) \end{aligned}$$

where, in the last line, all alpha-profiles of all spikes of all synapses have been indexed with $l = (\diamond, n, j)$, while $g_l = \bar{G}_j^\diamond/C$ for some (\diamond, j) , τ_l for some (\diamond) , and $t_l = \delta t_j + t_j^n$ for some (n, j) .

This yields, $\tilde{\omega}$ being fixed:

$$\begin{aligned} 0 &\leq \alpha^l(t - t_l) \leq \frac{1}{e} \\ 0 < \frac{1}{\tau_L} &\leq g(t)/C \leq \frac{1}{\tau_M} \stackrel{\text{def}}{=} \frac{1}{\tau_L} + \frac{1}{e} \sum_l g_l \quad (19) \\ \frac{t'-t}{\tau_L} &\leq \int_t^{t'} g(s)/C ds \leq \frac{t'-t}{\tau_M} \\ e^{-\frac{t'-t}{\tau_M}} &\leq \nu(t, t') \leq e^{-\frac{t'-t}{\tau_L}} \end{aligned}$$

On the other hand:

$$\begin{aligned}
i(t, \tilde{\omega})/C &= E_L \frac{1}{\tau_L} + E_+ \sum_{j=1}^N G_j^+(t, \tilde{\omega})/C + E_- \sum_{j=1}^N G_j^-(t, \tilde{\omega})/C \\
&\quad + E_\bullet \sum_{j=1}^N G_j^\bullet(t, \tilde{\omega})/C + I(\tilde{\omega})/C \\
&= E_L \frac{1}{\tau_L} + \sum_{\diamond=\{+, -, \bullet\}} E_\diamond \sum_{j=1}^N \tilde{G}_j^\diamond/C \sum_{n=1}^{M_j(t, \tilde{\omega})} \alpha^\diamond(t - \delta t_j - t_j^n) + I(\tilde{\omega})/C \\
&= i_0 + \sum_l E_l g_l \alpha^l(t - t_l)
\end{aligned} \tag{20}$$

with $I(\tilde{\omega}) = I^{(ion)}(\tilde{\omega}) + I^{(adp)}(\tilde{\omega}) + I^{(ext)}(\tilde{\omega})$ writing $i_0 = E_L \frac{1}{\tau_L} + I(\tilde{\omega})/C$ and using the same indexing mechanism with l .

A few algebra combining (19) and (20) leads to:

$$\begin{aligned}
\int_{t_0}^t \nu(s, t) \max(0, i(s)/C) ds &\leq \int_{t_0}^t e^{-\frac{t-s}{\tau_L}} \max(0, i_0 + \sum_l E_l g_l \alpha^l(s - t_l)) ds \\
&\leq \tau_L \left(1 - e^{-\frac{t-t_0}{\tau_L}}\right) \max(0, i_0) + \\
&\quad \sum_l \max(0, E_l) g_l \frac{\tau_l'}{\tau_l} e^{-\frac{t_0-t_l}{\tau_l}} \left[(t_0 - t_l') \left(e^{-\frac{t-t_0}{\tau_L}} - e^{-\frac{t-t_0}{\tau_l}} \right) - (t - t_0) e^{-\frac{t-t_0}{\tau_l}} \right]
\end{aligned}$$

writing

$$\begin{aligned}
\frac{1}{\tau_l'} &= \frac{1}{\tau_l} - \frac{1}{\tau_L} \\
t_l' &= t_l - \tau_l'
\end{aligned}$$

and on the other hand:

$$\nu(t_0, t) V(t_0) \leq \begin{cases} e^{-\frac{t-t_0}{\tau_L}} V(t_0) & \text{if } V(t_0) \geq 0 \\ e^{-\frac{t-t_0}{\tau_M}} V(t_0) & \text{if } V(t_0) \leq 0. \end{cases}$$

yielding for $t > t_0$:

$$\begin{aligned}
V(t) &\leq V(t_0) + \sum_{\diamond=\{L, M, +, -, \bullet\}} A_\diamond \left(1 - e^{-\frac{t-t_0}{\tau_\diamond}}\right) + \sum_{\diamond=\{+, -, \bullet\}} B_\diamond \alpha^\diamond(t - t_0) \\
&\leq V(t_0) + \left[\sum_{\diamond=\{L, M, +, -, \bullet\}, A_\diamond > 0} \frac{A_\diamond}{\tau_\diamond} + \sum_{\diamond=\{+, -, \bullet\}, B_\diamond > 0} \frac{B_\diamond}{\tau_\diamond} \right] (t - t_0)
\end{aligned} \tag{21}$$

since for $t > 0$, $(1 - e^{-t}) < t$ and $\alpha(t) < t$ are concave profiles as detailed in Figure 12. Here, A_\diamond and B_\diamond are not made explicit, but they are obvious to derive from the previous formulas.

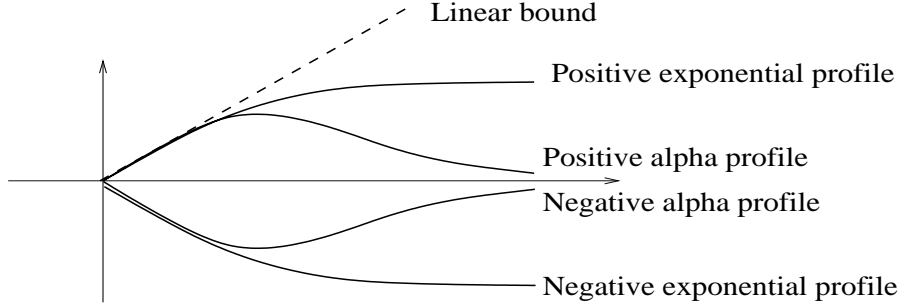


Figure 12: Bounding exponential and alpha profiles: positive profiles are concave and below the tangent at the origin (drawn in dashed), while negative profiles are convex and can only be bound by 0.

Computational complexity of the lower bound

Let us get into the calculation details and make (18) and (20) more explicit:

$$\begin{aligned}
g(t, \tilde{\omega})/C &= \frac{1}{\tau_L} + \sum_{\diamond=\{+,-\}} g^\diamond(t-t_0) \\
i(t, \tilde{\omega})/C &= i_0 + \sum_{\diamond=\{+,-,\bullet\}} E_\diamond g^\diamond(t-t_0)
\end{aligned} \tag{22}$$

writing

$$g^\diamond(t-t_0) = g_0^\diamond + g_1^\diamond(t-t_0) + (g_2^\diamond + g_3^\diamond(t-t_0))e^{-\frac{t-t_0}{\tau^\diamond}}$$

where g_i^\diamond are defined at some intermediate time t_0 , while $g'_i{}^\diamond$ are defined at some intermediate time $t'_0 < t_0$ yielding:

$$\begin{aligned}
g_0^\diamond &= \sum_{j=1}^N \bar{G}_j^\diamond / C \sum_{n=1}^{M_j(t, \tilde{\omega})} \frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond} e^{-\frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond}} \\
&= e^{-\frac{t_0 - t'_0}{\tau^\diamond}} (g_1'^\diamond (t_0 - t'_0) + g_0'^\diamond) + \sum_{t'_0 < t_j^n < t_0} \bar{G}_j^\diamond / C \frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond} e^{-\frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond}} \\
g_1^\diamond &= \sum_{j=1}^N \bar{G}_j^\diamond / C \sum_{n=1}^{M_j(t, \tilde{\omega})} \frac{1}{\tau^\diamond} e^{-\frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond}} \\
&= e^{-\frac{t_0 - t'_0}{\tau^\diamond}} g_1'^\diamond + \sum_{t'_0 < t_j^n < t_0} \bar{G}_j^\diamond / C \frac{1}{\tau^\diamond} e^{-\frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond}} \\
g_2^\diamond &= \sum_{j=1}^N \bar{G}_j^\diamond / C \sum_{n=1}^{M_j(t, \tilde{\omega})} \frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond} \\
&= g_2'^\diamond + (t_0 - t'_0) g_3'^\diamond + \sum_{t'_0 < t_j^n < t_0} \bar{G}_j^\diamond / C \frac{t_0 - \delta t_j - t_j^n}{\tau^\diamond} \\
g_3^\diamond &= \sum_{j=1}^N \bar{G}_j^\diamond / C M_j(t, \tilde{\omega}) \frac{1}{\tau^\diamond} \\
&= g_3'^\diamond + \sum_{t'_0 < t_j^n < t_0} \bar{G}_j^\diamond / C \frac{1}{\tau^\diamond}
\end{aligned} \tag{23}$$

Here $M_j(t, \tilde{\omega})$ is the number of spikes emitted by j at time $t - \delta t_j$ and received at time t . The key points regarding (23) are the following:

Recursive implementation Given the estimation of g_i^{\diamond} at some time $t'_0 < t_0$, we obtain g_i^{\diamond} at time t_0 (and thus compute quantities in (22) for any intermediate time t_0), using recursive formulas made explicit in (23) thus computing *once* the spike contribution for spike times $t'_0 < t_j^n < t_0$. In other words the calculation complexity is not the product of the number of spikes and the number of sampling steps, only a linear combination of them, drastically reducing the computational complexity for the simulation.

Numerical stability Usually, in recursive implementations errors accumulate.

Here, for g_0^{\diamond} and g_1^{\diamond} since we multiply by a factor $e^{-\frac{t_0 - t'_0}{\tau^{\diamond}}} < 1$ previous errors exponentially vanish with time, yielding the expected numerical stability. A step further, g_2^{\diamond} and g_3^{\diamond} are not subject to cumulative errors because they are simple sums. This numerical stability is reinforced by the fact that after a delay of $10\tau^{\diamond}$ the relative contribution of each spike vanishes, eliminating the related cumulative round-off errors⁷.

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⁷Note that the conductance is not reset by the occurrence of a spike though the membrane potential is reset. This induces a strong memory effect and history dependent evolution which is the core of gIF model complexity. However, the characteristic times τ^{\diamond} introduce somehow a natural time cut off allowing to simplify the numerical computation. More precisely, since:

$$\max_{t > 10\tau^{\diamond}} |\alpha^{\diamond}(t)| < 0.001 \quad \text{and} \quad \max_{t > 10\tau^{\diamond}} \left| \int_t^{+\infty} \alpha^{\diamond}(s) \right| < 0.001$$

we may neglect spike's evoked potential as soon as $t > 10\tau^{\diamond} + t_j^n$. This means that for an average firing rate of ρ spike/s the terms in (22) have about $10\tau^{\diamond}\rho N$ non negligible terms.

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